Brief Communication

Growth hormone in male infertility

Navneet Magon, Suneeta Singh[1], Ajit Saxena[2], Rakesh Sahay[3]

Department of Obstetrics and, Gynecology, Air Force Hospital, Kanpur, [1]Department of Obstetrics and Gynecology, Military Hospital, Mhow, Madhya Pradesh, [2]Department of Urology and Andrology, Indraprastha Apollo Hospitals, Delhi, [3]Department of Endocrinology, Osmania Medical College and Osmania General Hospital, Hyderabad, Andhra Pradesh, India

ABSTRACT

Growth hormone (GH) is expressed in a variety of tissues, including the testes, and has autocrine and paracrine functions as well. This, along with other factors, exerts autocrine and paracrine control over spermatogenesis. GH, used as an adjuvant therapy, induces spermatogenesis in non-responder patients with hypogonadotropic hypogonadism, who are not responding to gonadotropin or pulsatile luteinizing hormone (LH) therapy. GH has an important physiological role to play in spermatogenesis and male fertility.

Key words: Growth hormone, infertility, male, pituitary, spermatogenesis

Growth hormone (GH) expression is not limited to the pituitary, neither is its function limited to simple endocrine effects on growth. GH is expressed in a variety of tissues, including the testes, and has autocrine and paracrine functions as well.

The process of spermatogenesis is essential for human reproduction. A simple sounding process is mediated by a variety of factors, including multiple hormonal influences. The gonadotropin-releasing hormone (GnRH), LH, Follicle-stimulating hormone (FSH), and testosterone all play an important role in the development and maturation of sperms. At the same time, various locally secreted peptides and proteins such as GH, IGF-1, cytokines, activin, inhibin, follistatin, and estrogen, exert autocrine and paracrine control over spermatogenesis.[1]

The growth hormone acts directly and indirectly via hepatic IGF-1, at the testicular level, to promote sperm production. The locally produced GH may act in a paracrine or autocrine fashion to regulate local processes that are strategically regulated by pituitary GH. GH promotes early development of spermatogonia, and ensures complete maturation as well.

Growth hormone–deficient men have small-sized testes. GH has been found to be deficient in phenotypically normal, azoospermic men, with maturation arrest, a finding confirmed by clonidine stimulation tests.[2] Conversely, the sperm count is low or nil in men with GH deficiency. GH resistance in men is also associated with reduced fertility.[3]

Growth hormone restores sperm concentration, morphology, and motility in GH-deficient rats[4] as well as men. GH, used as adjuvant therapy, induces spermatogenesis in non-responder patients with hypogonadotropic hypogonadism, who are not responding to gonadotropin or pulsatile LH therapy. A study on nine oligozoospermic and nine asthenozoospermic men treated with GH for 12 weeks reported increased sperm motility in both groups, and three pregnancies were reported in asthenozoospermia, but not in oligozoospermia.[5]

An Indian prospective, open-label, non-randomized observational study of 14 men, aged between 26 and 35 years, with normogonadotropic idiopathic oligoasthenospermia has described the beneficial effects of growth hormone 1.5 IU / day, administered for six months. Semen volume,

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Corresponding Author: Dr. Navneet Magon, Obstetrician, Gynecologist and Endoscopic Surgeon, Department of Obstetrics and Gynecology, Air Force Hospital, Kanpur, Uttar Pradesh, India. E-mail: navneetmagon@gmail.com

count, and motility were improved in all patients. The increase was most marked during the first three months of therapy. Not much improvement was noticed during the latter half of the treatment. None of the patients experienced any side effects. Three subjects fathered children over the next one year, two with the help of intrauterine insemination.[6]

At the same time, other authors have also reported lack of beneficial effect with this therapy.[7] Therefore, GH cannot be promoted as a panacea for all subfertile men.

The GH and recombinant human insulin-like growth factor-I (rhIGF-I) can be utilized in improving the outcome of IVF as well. These drugs have been reported to maintain sperm motility longer after a 24-hour treatment at room temperature in mature equine spermatozoa, without any deletiroius effects. This property can be utilized to store spermatozoa longer at room temperature in Assisted Reproductive Technology (ART) centres.[8]

In conclusion, GH has an important physiological role to play in spermatogenesis and male fertility. More studies are required to define the exact place of GH therapy in clinical practice. Although it certainly merits a trial in GH-deficient patients, it may be used in non-responding normogonadotropic idiopathic oligoasthenospermia. A close watch must be kept for metabolic side effects. Its true potential will be realized only if endocrinologists: both medical and reproductive, team together to analyze the patient populations and where it can be used.

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